

# Characterization of Focal Atrial Tachycardia Using High-Density Mapping

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<b>OBJECTIVES</b>	The goal of this study was to characterize the origin of focal atrial tachycardias (AT).
<b>BACKGROUND</b>	Focal ATs originate from a small area and spread centrifugally; however, activation at the AT origin has not been characterized.
<b>METHODS</b>	Twenty patients with AT having failed prior ablation or occurring after atrial fibrillation ablation were studied. After excluding macro-re-entry, AT was mapped using a 20-pole catheter (five radiating spines; diameter 3.5 cm), performing vector mapping to identify the earliest activity followed by high-density mapping at the AT origin. Localized re-entry was considered if >85% of the tachycardia cycle length (CL) was observed within the mapping field and was confirmed by entrainment.
<b>RESULTS</b>	A total of 27 ATs were mapped to the pulmonary vein ostia (n = 5), and left (n = 16) and right atria (n = 6). A localized focus was evidenced at the site of origin in 19 ATs (70%), whereas in 8 (30%), localized re-entry was evidenced by $95.2 \pm 4.5\%$ of the tachycardia CL recorded within the mapping field and entrainment showed a post-pacing interval <20 ms longer than tachycardia CL (6 of 6 tested). Localized re-entry had a shorter CL ( $p = 0.009$ ), slowed conduction at its origin (fractionated potential $115 \pm 19$ ms vs. $64 \pm 22$ ms, representing $49 \pm 10\%$ and $20 \pm 10\%$ of tachycardia CL, respectively; $p < 0.0001$ ), and were more often contiguous with regions of electrical silence or conduction abnormalities (88% vs. 32%; $p = 0.01$ ). In addition, mapping documented varying degrees of intra-atrial conduction block, preferential conduction (n = 5), and rapid bursts of myocardial activity (n = 1). At $11 \pm 7$ months, none have had recurrence of AT.
<b>CONCLUSIONS</b>	High-density multielectrode mapping can be used to perform vector mapping to localize complex AT. It provides novel insight into the mechanisms of focal AT, distinguishing focal AT from localized re-entry. (J Am Coll Cardiol 2005;46:2088–99) © 2005 by the American College of Cardiology Foundation

Focal atrial tachycardias (AT) are characterized by atrial activity that originates from a small area (focus) and spreads out centrifugally (1). The mechanisms of these arrhythmias have been the subject of debate, with increased automaticity, triggered activity, and micro-re-entry all considered possibilities. Atrial tachycardias demonstrate an anatomic predilection; in the right atrium (RA), along the crista terminalis, tricuspid annulus, coronary sinus (CS) ostium, and the septum (2–5), and in the left atrium (LA), from the septum, mitral annulus, and pulmonary vein (PV) ostia (6–8). Characterization of the electrocardiographic and electrophysiologic features of tachycardia from these sites has facilitated their mapping and ablation (2,4–9).

Nevertheless, in 5% to 24% mapping is prolonged and can result in failure of ablation, particularly in patients with previous atrial surgery or ablation, or when tachycardia arises from a less well characterized region (10). Despite the frequent use of multielectrode catheters and three-dimensional mapping systems, limited information is available regarding the localized activation at the tachycardia origin. The aim of the present study was to evaluate the role of localized high-density mapping to identify and characterize the origin of focal AT.

## METHODS

**Study population.** The study comprised 20 patients undergoing ablation of AT. Patients were selected on the basis of having failed prior attempts at AT ablation (n = 8) or presenting with regular arrhythmia that were not macro-reentrant after ablation for atrial fibrillation (AF) (n = 12). Baseline characteristics of these patients are presented in Table 1. All patients gave written informed consent to the study, which was approved by the institutional clinical research and ethics committee.

**Electrophysiology study.** Electrophysiological study was performed with the minimal use of sedation. All antiar-

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#### Abbreviations and Acronyms

AF = atrial fibrillation  
AT = atrial tachycardia  
CI = confidence interval  
CL = cycle length  
CS = coronary sinus  
LA = left atrial/atrium  
PV = pulmonary vein  
RA = right atrial/atrium

rhythmic drugs, with the exception of amiodarone, were ceased  $\geq 5$  half-lives before the procedure.

Surface electrocardiogram and bipolar endocardial electrograms were continuously monitored and stored on a computer-based digital amplifier/recorder system (Bard Electrophysiology, Lowell, Massachusetts). Intracardiac electrograms were filtered from 30 to 500 Hz and measured at a sweep speed of 100 to 200 mm/s.

**CONVENTIONAL MAPPING.** In the absence of spontaneous tachycardia, induction was attempted by burst pacing and programmed extrastimuli. If these were unsuccessful, isoproterenol was infused (1 to 6  $\mu\text{g}/\text{min}$ ). Standard electrophysiologic criteria were used to diagnose AT (11). Initial mapping was performed to exclude patients with a macro-reentrant mechanism. This was performed by positioning a quadripolar catheter (Xtrem, Ela Medical, Montrouge, France) in the CS and using a roving 4-mm tip quadripolar ablation catheter (Biosense-Webster, Diamond Bar, California/Bard Electrophysiology) to perform activation and entrainment mapping. If mapping failed to demonstrate macro-re-entry within the RA, mapping of the LA was performed. After LA access, heparin (0.5 mg/kg) was administered. Macro-re-entry was excluded by the following criteria: 1) sequential activation mapping demonstrating  $< 50\%$  of the tachycardia cycle length (CL); and 2) a post-pacing interval at  $\geq 3$  sites/atria being  $\geq 40$  ms longer than the tachycardia CL.

**HIGH-DENSITY MAPPING CATHETER.** After excluding macro-re-entry, mapping was performed utilizing a new high-

density mapping catheter (PENTARAY, Biosense-Webster). This 7-F steerable catheter ( $180^\circ$  of unidirectional flexion) has 20 electrodes distributed over five soft radiating spines (1-mm electrodes separated by 4-4-4 or 2-6-2 mm inter-electrode spacing) allowing splaying of the catheter to cover a surface diameter of 3.5 cm. The spines have been nominated alphabetical nomenclature (A to E) with spines A and B being recognized by radio-opaque markers (Fig. 1A).

**VECTOR MAPPING TO LOCALIZE THE ORIGIN OF TACHYCARDIA.** Localization of the atrial focus was performed during tachycardia or atrial ectopy. Guided by the electrocardiographic appearance and previous mapping information, the catheter was sequentially applied to the endocardial surface in various atrial regions to allow rapid activation mapping. Mapping was performed to identify the earliest endocardial activity relative to the P-wave and/or to a fixed catheter positioned within the CS. By identifying the earliest site of activation around the circumference of the high-density catheter, vector mapping was performed, moving the catheter and applying it to the endocardium in the direction of earliest activation (outer bipoles) to identify the tachycardia origin and bracket activation, that is, demonstrating later activation in all surrounding regions (Fig. 1B).

Atrial tachycardia was considered to be localized re-entry if the mapping field demonstrated  $> 85\%$  of the tachycardia CL at the site of earliest activation, and was confirmed by entrainment.

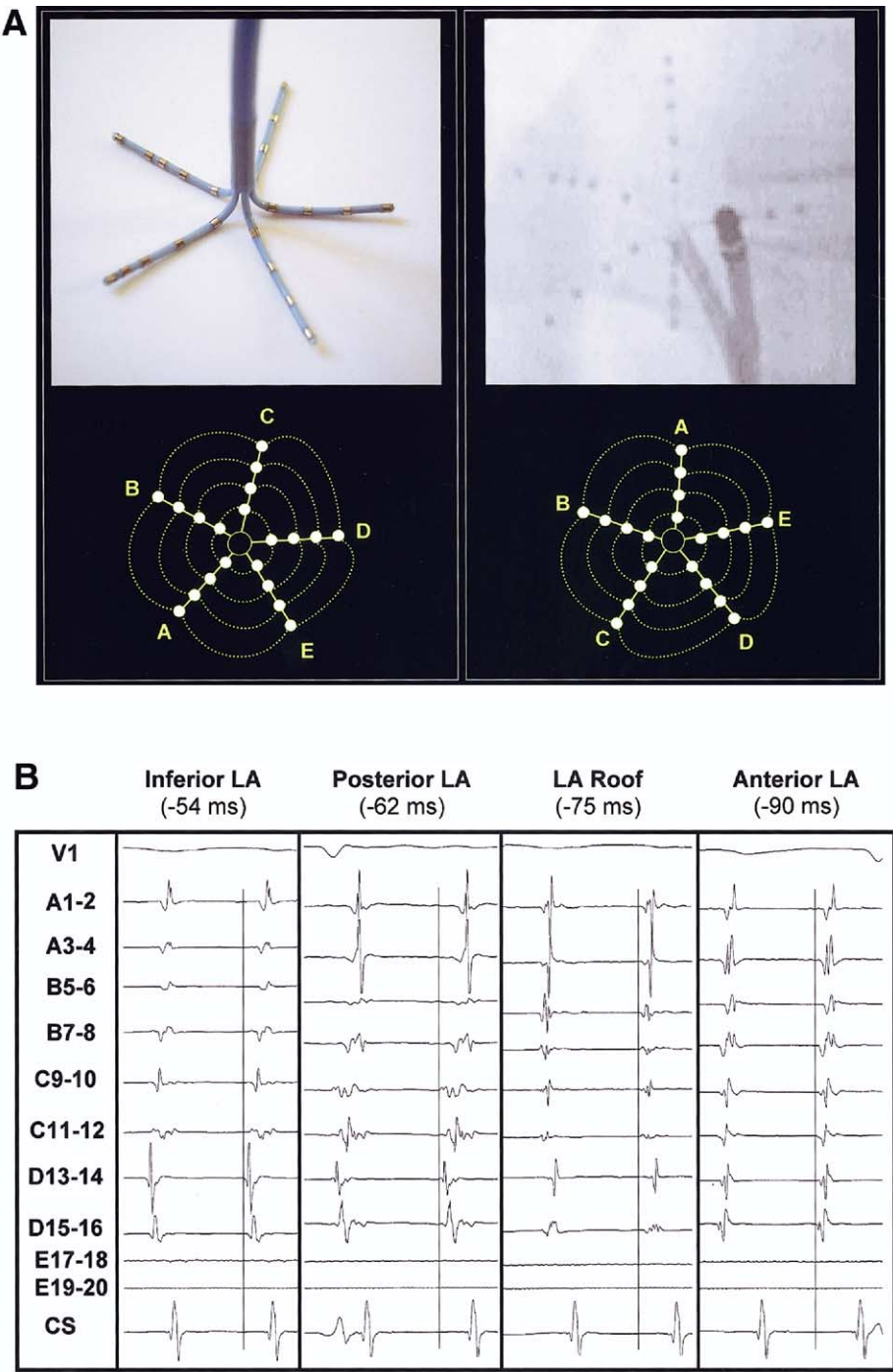
**Radiofrequency ablation.** The accuracy of mapping was confirmed by the termination or change in tachycardia with ablation. A change in tachycardia was defined by a change in the P-wave morphology with an alteration of the endocardial activation. Ablation was performed at the site of earliest endocardial activation relative to the P-wave when tachycardia was bracketed and at the most fractionated site when the entire tachycardia CL was identified. Ablation used continuous temperature feedback control of power output to achieve a target temperature of  $50^\circ\text{C}$  to  $60^\circ\text{C}$ , for a delivered power of 30 to 50 W. The acute procedural success was defined by the absence of tachycardia or ectopy in the 30

**Table 1.** Patient Characteristics

	Atrial Tachycardia Without AF (n = 8)	Atrial Tachycardia After AF Ablation (n = 12)	p Value
Age (yrs)	36 $\pm$ 18	54 $\pm$ 11	0.02*
Male gender (no)	5 (63%)	10 (83%)	0.3†
AT duration (months)	32.5 $\pm$ 39.7 (range 3–120)	2.4 $\pm$ 1.5 (range 1–6)	0.0005‡
Permanent/paroxysmal AF	—	6/6	—
Structural heart disease	4	0	0.02†
LV dysfunction	4	—	—
Mitral valve disease	1	—	—
Left atrial size (mm)	42 $\pm$ 9	49 $\pm$ 7	0.05*
LV ejection fraction (%)	52 $\pm$ 20	67 $\pm$ 8	0.1*

\*Student t test; †Fisher exact test; ‡Wilcoxon rank sum test.

AF = atrial fibrillation; AT = atrial tachycardia; LV = left ventricular.



**Figure 1.** (A) Shows a picture and a fluoroscopic image of the high-density mapping catheter. Below each figure is a schematic representation indicating the orientation of the catheter spines. Note the marker band on spine-A is between electrodes 1 to 2, and on spine-B between electrodes 2 to 3. (B) Demonstrates vector mapping to identify the earliest site of activation. Shown is the catheter in four distinct locations within the left atrium (LA). Mapping is commenced along the inferior LA where the earliest activation is 54 ms ahead of the coronary sinus (CS). The catheter is moved in the direction of the spine demonstrating the earliest activation (spine-D). In the mid-posterior LA, activation precedes the CS by 62 ms. Again the catheter is moved in the earliest direction (spine-D) to a more cranial location on the LA roof. At this site, activation precedes the CS by 75 ms. At this site, spine-B, which is slightly anterior, is the earliest. Moving the catheter in the direction of spine-B to the anterior-superior LA demonstrates the site with the earliest endocardial activation (90 ms ahead of the CS, which was 40 ms ahead of the P-wave).

min after ablation despite infusion of isoproterenol and burst atrial pacing.  
**Statistical analysis.** Continuous variables are reported as mean  $\pm$  SD or median and range. Comparison between

groups was performed with the Student *t* test or when data was not normally distributed (Shapiro-Wilk test), the Wilcoxon rank sum test. Categorical variables are reported as number and percentage, and compared using

the Fisher exact test. Statistical significance was established at  $p < 0.05$ .

## RESULTS

**Patient characteristics.** Table 1 shows the baseline characteristics according to the presence of prior AF ablation. Patients with AT and no AF were younger ( $p = 0.02$ ; Student  $t$  test) and had smaller atrial chambers ( $p = 0.05$ ; Student  $t$  test). They had AT for significantly longer durations ( $p = 0.0005$ ; Wilcoxon rank sum test). All patients with AF had undergone PV electrical isolation and additional linear ablation as previously described (12–14). These patients presented with AT  $3.1 \pm 2.1$  months after AF ablation.

**Localization of AT.** Twenty-seven different ATs were mapped and ablated during repetitive bursts or sustained AT (median CL 262 ms, range 190 to 550 ms, 95% confidence interval [CI] 246 to 425 ms). In 18 patients a single tachycardia was ablated and in 2 patients, 1 without AF and 1 after AF ablation; AT originated from six and three separate sites, respectively. Figure 2 demonstrates a schematic representation of the AT origin. In patients with AT without AF, the tachycardia origin was localized to the PV ostia ( $n = 3$ ), anterior LA ( $n = 4$ ), posterior or roof of the LA ( $n = 2$ ), LA appendage ( $n = 1$ ), RA appendage ( $n = 2$ ), and high-lateral RA ( $n = 1$ ). One focus located along the anterior LA and one in the high-lateral RA were contiguous with regions of spontaneous electrical silence. In patients with AT after AF ablation, the tachycardia origin was localized to the PV ostia ( $n = 2$ ), anterior or septal LA ( $n = 5$ ), posterior LA or roof ( $n = 3$ ), inferior LA ( $n = 1$ ),

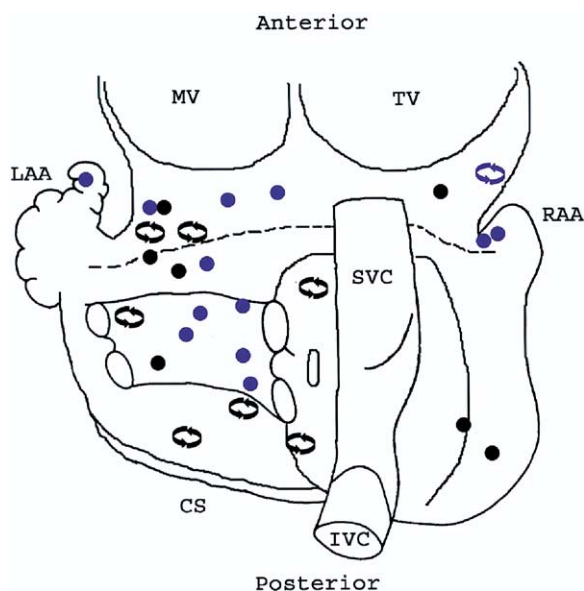
posterior RA or crista terminalis ( $n = 2$ ), and anterior RA ( $n = 1$ ); 9 of 14 ATs occurred in close proximity to previous ablations and a further 2 arose from regions demonstrating marked conduction abnormality with spontaneous electrical silence (scar) or double potentials along the posterior RA and the anterior LA, respectively.

All ATs were successfully ablated with  $7.6 \pm 4.8$  min (duration to eliminate and consolidate) of radiofrequency energy application per site. This was performed with fluoroscopic and procedural durations of  $41.2 \pm 19.4$  min and  $135.9 \pm 36.2$  min, respectively. No complications were observed with the use of the multispine catheter.

In two patients with AT after AF ablation, AT recurred one and two days after ablation, respectively, and re-ablation was performed successfully at the same sites as the initial ablation. At  $11 \pm 7$  months after the last procedure, one patient has required further ablation for AF, but there have been no further recurrences of AT.

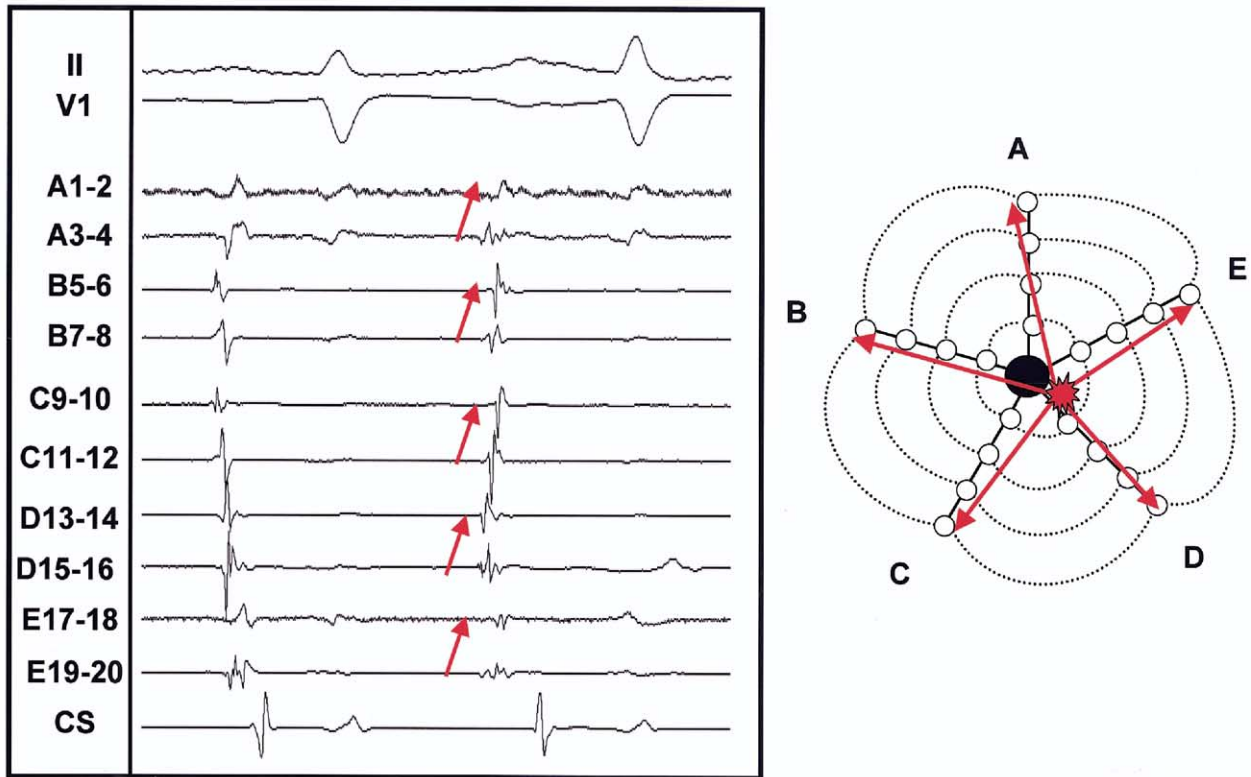
**Focal AT versus localized re-entry.** In all 27 ATs mapped in this study, atrial activation was observed to be centrifugal arising from a distinct region of the atria. In 19 (70%, 95% CI 50% to 86%), the origin of the AT demonstrated radial activation from a focal site (Fig. 1B), including 5 with activation commencing from the center of the mapping field (Fig. 3). Such focal AT demonstrated earliest activation  $48.5 \pm 24.7$  ms (range 16 to 98 ms, 95% CI 37.4 to 59.6 ms) ahead of the P-wave, and the mapping catheter recorded the first  $94.0 \pm 37.0$  ms of tachycardia (range 40 to 170 ms, 95% CI 76.8 to 111.2 ms) representing  $26.1 \pm 16.1\%$  of the tachycardia CL (range 7.7% to 69.4%). Ablation at this site resulted in AT termination in all cases.

In the remaining eight ATs (30%, 95% CI 14% to 50%), mapping at the site of origin demonstrated  $95.2 \pm 4.5\%$  (95% CI 92.1% to 98.4%) of the tachycardia CL (Figs. 4 to 6). Four of these patients required programmed extrastimuli to induce arrhythmia, while in the remaining, AT occurred spontaneously. In six, entrainment could be performed from one to three sites within this region and demonstrated a post-pacing interval that was  $<20$  ms longer than tachycardia CL (Fig. 5). In the remaining two, entrainment could not be assessed due to inability to capture the atrium at this site. Seven of these ATs were observed in patients with a history of AF; four in proximity to previously ablated regions (Figs. 2 and 4) and three occurring along the anterior or septal LA, an area not previously targeted for ablation (Figs. 2 and 5). In all these patients, the site of origin demonstrated prolonged fractionated electrograms (Figs. 4 to 6). Such local re-entrant activation, displaying the entire tachycardia CL, was observed in only one patient in the absence of a history of AF. This 45-year-old man had no known structural heart disease or systemic ailments; however, during previous mapping (before ablation), he was observed to have a large area of spontaneous low voltage and electrical silence along the lateral RA (Fig. 6A). For the current study, the region of electrical silence could readily be identified with activation mapping localizing the AT to an



**Figure 2.** Schematic representation of the location of atrial tachycardias in this cohort. Each dot represents a focal tachycardia, and localized re-entry is represented by arrows (blue for those without and black for those with a history of atrial fibrillation). CS = coronary sinus; IVC = inferior vena cava; LAA = left atrial appendage; MV = mitral valve; RAA = right atrial appendage; SVC = superior vena cava; TV = tricuspid valve.





**Figure 3.** Anterior left atrial tachycardia focus at the center of the mapping catheter. Demonstrated are the electrograms and a schematic representation of activation. The catheter was positioned directly on the site of the tachycardia focus and resulted in a proximal-to-distal activation on four or five spines of the catheter. In this case, spine-D demonstrates the earliest activity and the smallest activation gradient of all spines. Ablation at this site terminated tachycardia. Note the much smaller relative activation time difference between electrodes on account of the localized mapping, compared to conventional activation mapping. CS = coronary sinus.

area cranial and contiguous with this region (Figs. 6B and 6C). While the entire tachycardia CL was recorded locally within the field of mapping (with centrifugal activation to the remaining atria), entrainment could not be performed due to the very low voltage amplitude (0.03 to 0.07 mV). In patients who demonstrated a localized re-entrant arrhythmia, ablation was performed at the site with the longest fractionated signal determined by the mapping catheter, which had an electrogram duration of  $114.5 \pm 18.6$  ms (range 88 to 150 ms, 95% CI 102 to 127 ms) representing  $48.9 \pm 10.2\%$  of the tachycardia CL. Ablation at this site terminated tachycardia in all.

Table 2 shows the distinctive features of localized re-entry compared to focal AT. Compared to patients with focal AT, patients with localized re-entry had a shorter tachycardia CL ( $p = 0.009$ ; Wilcoxon rank sum test), evidence of slowed conduction at the origin of tachycardia ( $p < 0.0001$ ; Student  $t$  test), tachycardia arose more often contiguous with regions of electrical silence or double potentials ( $p = 0.01$ ; Fisher exact test), and had a history of AF ( $p = 0.03$ ; Fisher exact test).

**Complex intra-atrial conduction.** Intra-atrial conduction block at the site of origin of AT was observed in five patients, one without previous AF ablation. The degree of intra-atrial conduction block observed was variable ranging from occasional concealed beats to regions demonstrating

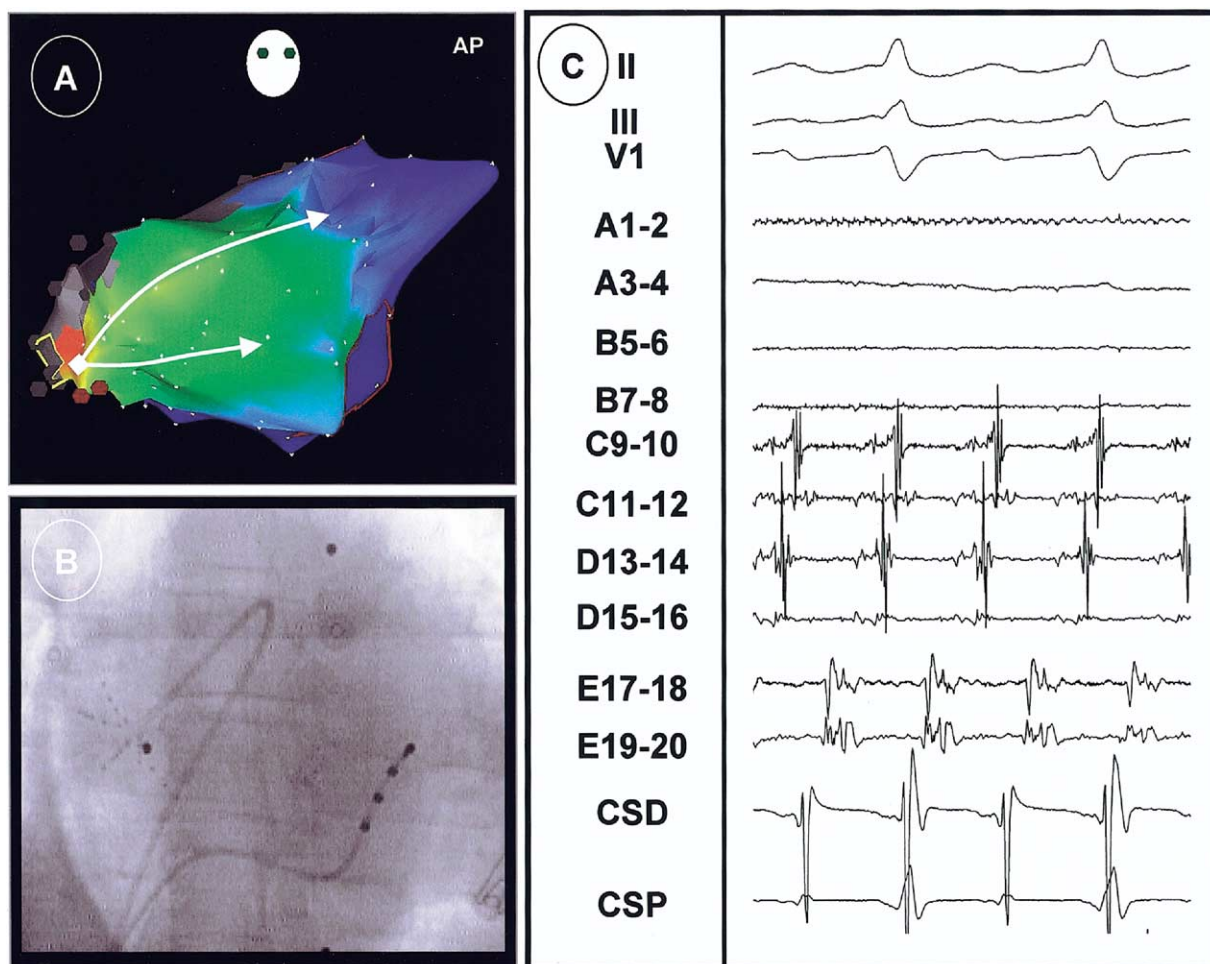
dissociated rhythms (Figs. 7A and 7B). In the same patient as in Figure 7A, during mapping of the AT, an area of electrical silence was observed in the low-lateral RA. This region, despite being surrounded by areas of electrical silence, contained a small pocket of atrial myocardium ( $0.5 \times 0.5$  cm) demonstrating high-frequency activity (mean CL 83 ms) recorded on a single 5-mm bipole (Fig. 7B). Ablation was performed at this site and locally eliminated all electrical activity but had no effect on the AT, suggesting that this island of tissue may have been sequestered from the remaining atria.

In addition, at the site of origin of the AT, preferential conduction was observed from origin-to-exit or breakout to the remaining atria in one patient (Fig. 8). In three further cases, earliest atrial activation preceded the P-wave by  $\geq 80$  ms with a gap in the locally mapped activation and a significant delay to the surrounding atria, presumably due to the presence of a preferential isthmus of conduction.

## DISCUSSION

This study presents new information regarding the feasibility of high-density contact mapping during clinical electrophysiological procedures and the mechanisms of "focal" AT.

First, it demonstrates the clinical utility of multielectrode localized mapping to perform vector mapping to rapidly



**Figure 4.** Localized re-entry as a mechanism of atrial tachycardia centrifugally activating the remaining atria. This example is in near proximity to the previous right inferior pulmonary vein (PV) ablation. (A) Represents activation mapping demonstrating centrifugal activation of the left atrium from an area immediately anterior to the still-isolated right-inferior PV. (B) Demonstrates the positioning of the high-density catheter in this region and the resultant activation sequence is shown in (C). Re-entry was confirmed by entrainment, and ablation successfully terminated tachycardia (brown tags, A). Note that spine-A and -B are within the isolated PV. CSD = distal coronary sinus; CSP = proximal coronary sinus.

identify the source of AT, particularly those localized to the LA and occurring after prior ineffective ablation.

Second, high-density mapping at the site of origin of tachycardia provided novel insights into the mechanisms of these apparently “focal” tachycardias that activated the atria in a centrifugal manner; in 30% the entire CL was identified within the localized mapping field with entrainment confirming a re-entrant mechanism.

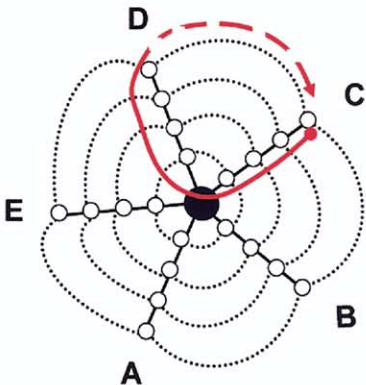
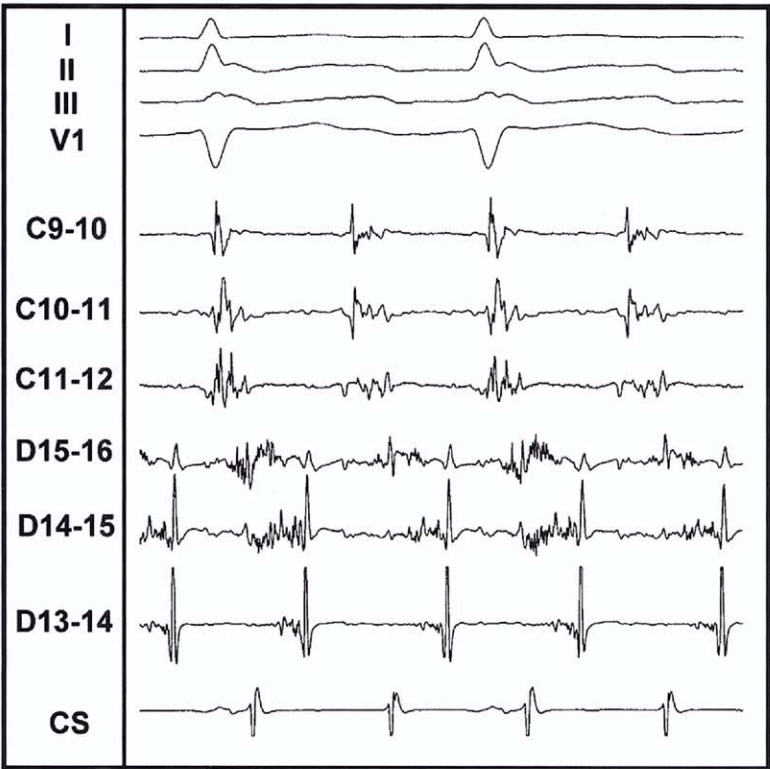
Finally, evidence of tiny regions of high-frequency atrial activity, and complex local and intra-atrial conduction block were observed to manifest in misleading electrocardiographic features.

**Prior studies mapping AT.** Atrial tachycardias demonstrate a characteristic anatomic predilection to endocardial structures in both the RA and LA (2–8). Knowledge of the endocardial anatomy, in concert with the specific features and activation patterns, have facilitated their mapping and ablation. While the association with atrial anatomy is observed in most cases, the mapping of AT arising from other sites is more difficult and is compounded by limited

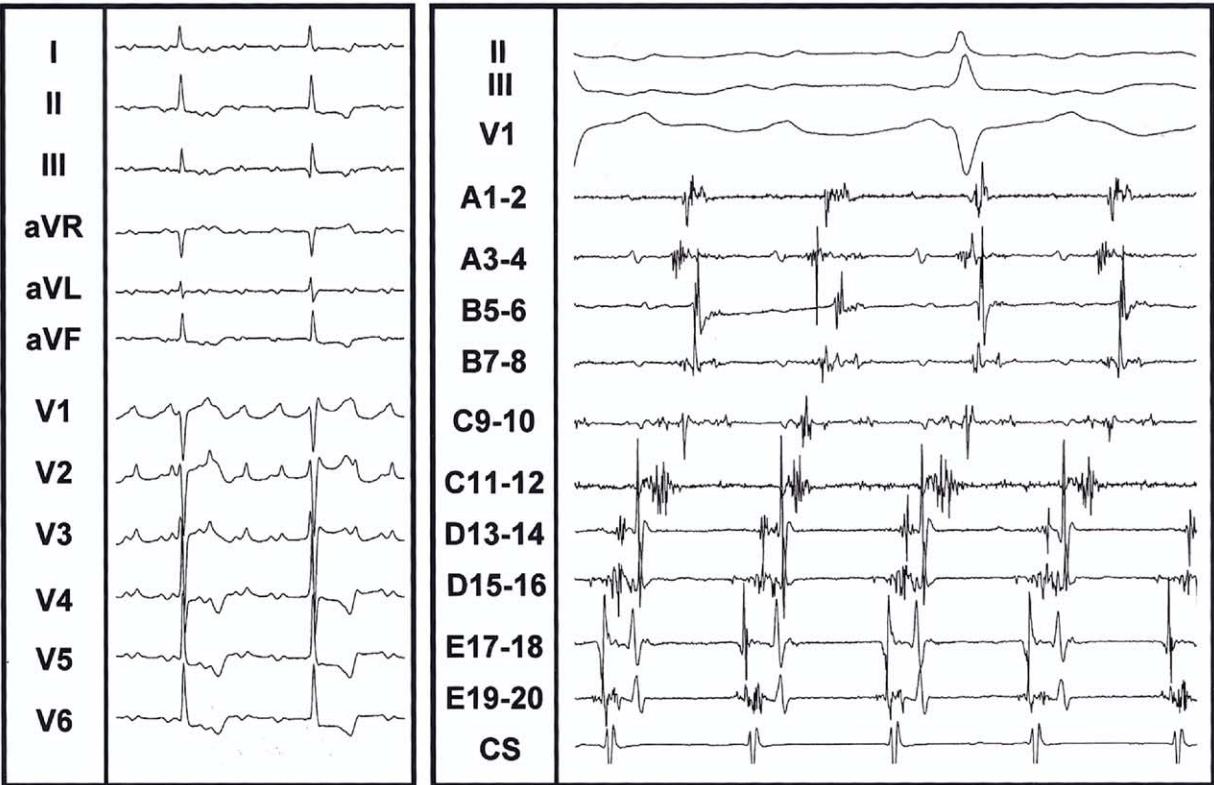
experience in mapping these less frequent arrhythmias. In addition, the incidence of such arrhythmias has burgeoned as techniques for the ablation of AF are applied to greater numbers of patients, further augmenting the need for mapping techniques for such sources that occur frequently within the LA. Although LA tachycardias may be recognized by the characteristic electrocardiogram or intracardiac activation sequence (9,15,16), subsequent localization of their origin requires detailed mapping, and reports suggest a greater prevalence of procedural failure (17).

Regular atrial arrhythmias have been reported variably after surgical or catheter ablation procedures for AF. Although patients undergoing atrial substrate ablation are more likely to develop these arrhythmias (13,14,18), they have also been described in the setting of PV ablation alone (19–21). The majority of these arrhythmias have a macro-reentrant mechanism, utilizing regions of incomplete or recovered ablation lesions to sustain arrhythmia. A smaller proportion of patients have a focal mechanism for their arrhythmias (18,21).

A

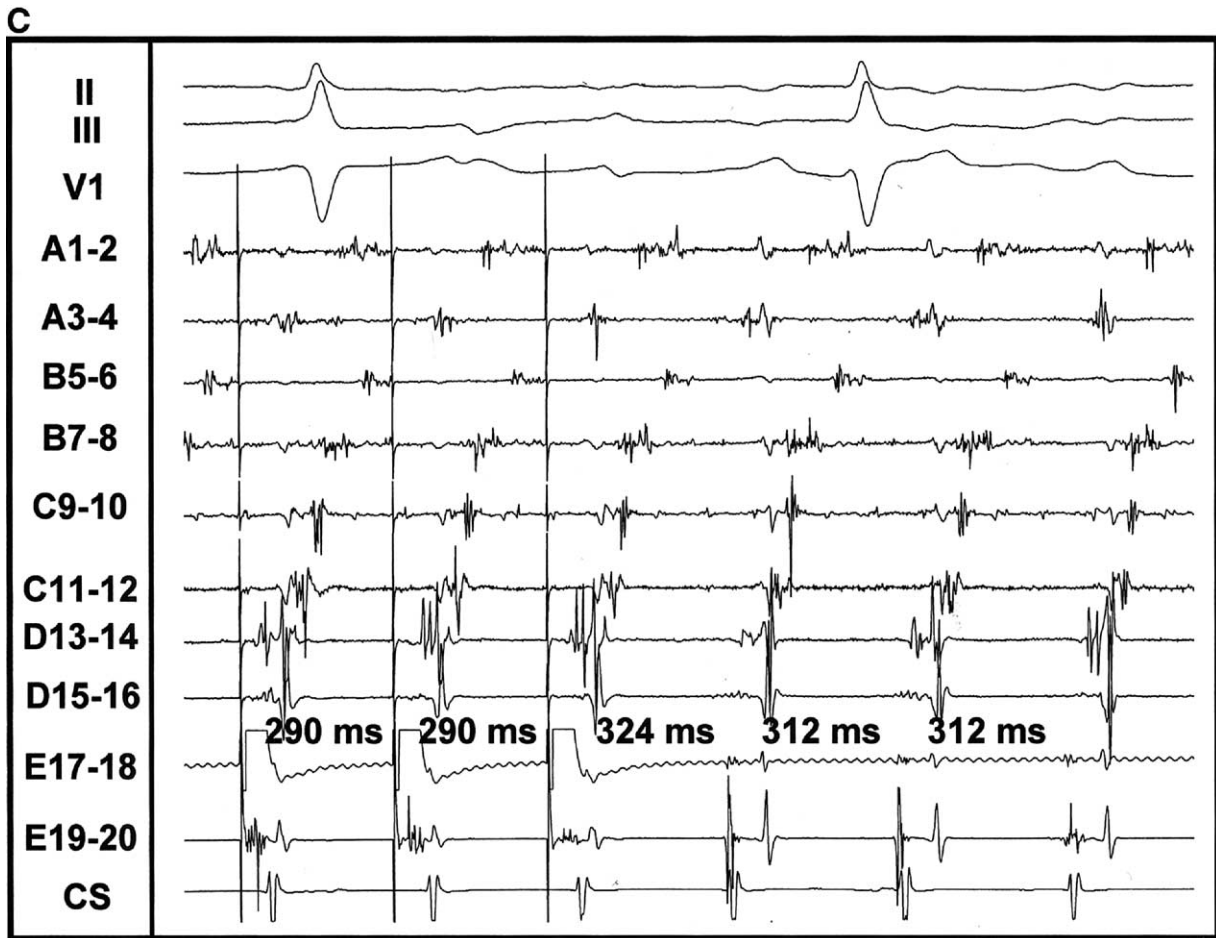


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**Figure 5.** Localized re-entry occurring at a site not in proximity to previous ablation. **(A)** Demonstrates tachycardia arising from the anterior-superior left atrial (LA) wall. Electrograms from only spine-C and -D have been presented to demonstrate the cascade of activation (spine-C is organized from distal-to-proximal and spine-D from proximal-to-distal). Note each spine in this case was configured to provide three bipoles. Also shown is a schematic representation of the activation cascade. Entrainment confirmed a re-entrant mechanism, and ablation terminated tachycardia. **(B and C)** Demonstrate activation and entrainment of tachycardia in a different patient. Tachycardia was localized on the anterior-septal LA wall. Entrainment demonstrates a post-pacing interval 12 ms longer than the tachycardia cycle length (CL). In this patient, entrainment could be performed from three sites in the mapping field and were as above. However, entrainment performed in the surrounding atria demonstrated post-pacing intervals  $\geq 40$  ms longer than tachycardia CL. CS = coronary sinus.

The mapping catheter used in the current study provided a localized density of mapping within the LA that has previously not been available. It allowed rapid activation mapping of the atria to facilitate identifying the tachycardia focus that was often localized to regions of conduction abnormalities. Whether these conduction abnormalities were pre-existing or a consequence of prior ablation is not known; however, in patients after AF ablation, AT originated from sites both in proximity to and in regions not previously targeted by ablation. Interestingly, while current classification of these arrhythmias suggests that they were "focal," demonstrating centrifugal activation of the atria, we observed that some had a re-entrant mechanism.

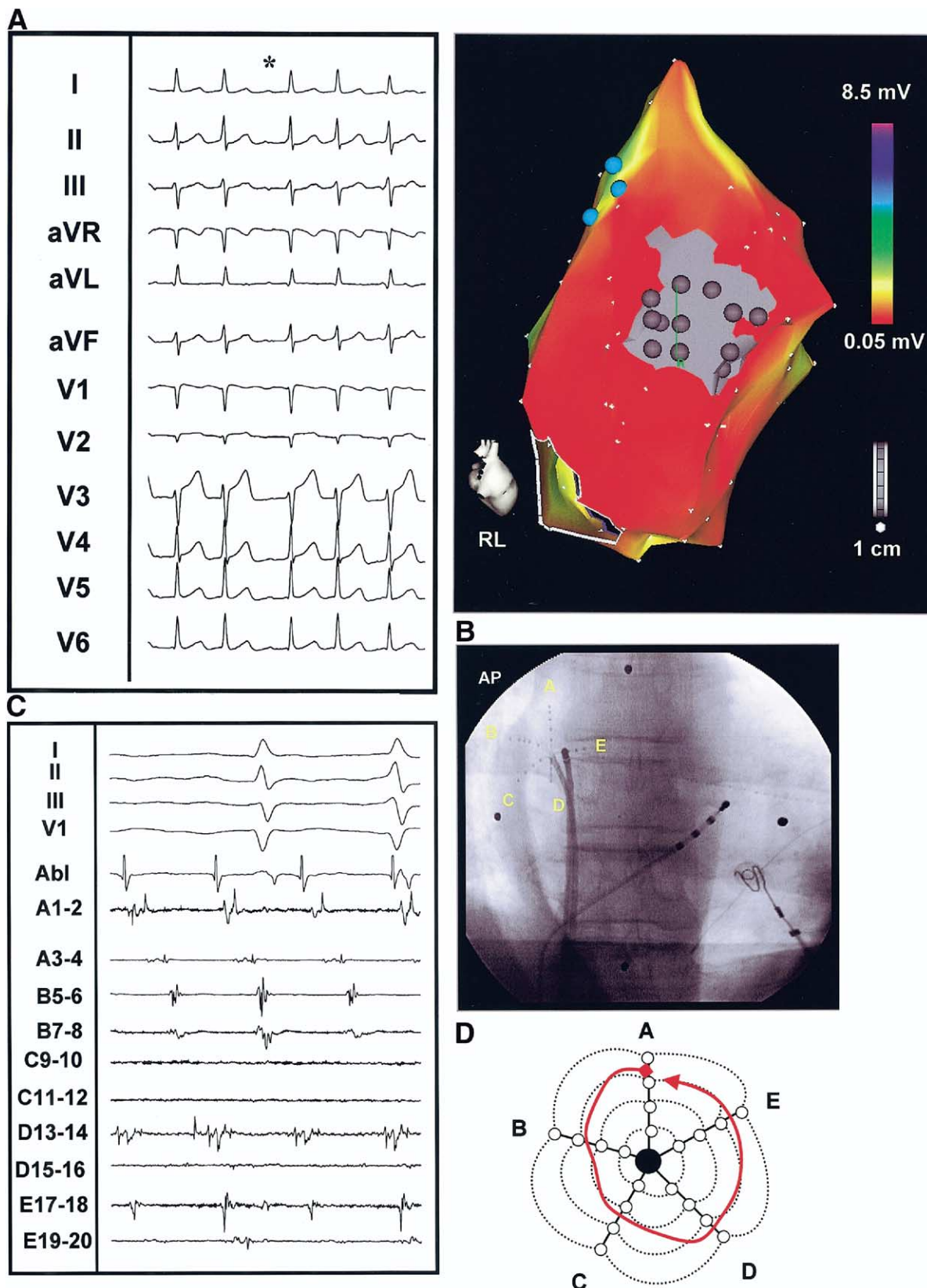
**Focal versus localized re-entry as the mechanism of AT.** Current consensus for the classification of AT suggest that a focal AT demonstrates atrial activation commencing from a localized region and spreading centrifugally (11). In contrast, macro-re-entry is defined by activation that can be recorded over the entire tachycardia CL around a "large"

central obstacle, which is generally several centimeters in diameter. These latter arrhythmias exhibit less CL variation, and entrainment maneuvers performed at sites separated by at least 2 cm are consistent with a re-entrant mechanism (11).

In the current cohort of patients, all of whom displayed centrifugal activation from a localized region, 30% demonstrated the entire tachycardia CL within the localized region at the AT origin. Half of these patients had arrhythmia induced by programmed extrastimuli, and a re-entrant mechanism was confirmed by entrainment. The local electrogram at the site of origin displayed prolonged fractionated signals, which were contiguous with regions of electrical silence. While this probably constitutes what has been referred to as micro-re-entry, these observations herald the need for further classifying these localized but re-entrant arrhythmias, which are likely to be increasingly recognized with improving mapping technologies.

**High-density mapping technologies for AT.** An expanding array of mapping technologies have been developed to





**Figure 6.** Localized re-entry in a patient without a history of atrial fibrillation. (A) Presents the electroanatomic voltage map (lateral right atrial en face) created during this patient's previous mapping procedure (before ablation) together with the electrocardiographic features of the tachycardia. Note the low-amplitude P-wave (\*). (B) shows the catheter position immediately superior to the region of electrical silence with (C) showing the resulting activation at the site of origin. The electrograms in this patient have not been arranged, but activity is observed throughout the tachycardia cycle length and is demonstrated visually in the schematic (D). In this patient, the atria could not be captured to perform entrainment from this site. AP = anterior-posterior; RL = right lateral.

**Table 2.** Focal AT Versus Localized Re-Entry

	Localized Re-entry (n = 8)	Focal AT (n = 19)	p Value
History of AF, n (%)	7 (88)	7 (37)	0.03*
TCL, median (range; ms)	246 (190–306)	424 (210–550)	0.009†
TCL in mapping field (%)	95.2 ± 4.5	26.2 ± 16.1	0.0001†
Electrogram at earliest site			
Duration (ms)	114.5 ± 18.6	63.7 ± 22.5	<0.0001‡
% TCL	48.9 ± 10.2	19.7 ± 9.7	<0.0001‡
Contiguous scar/double potentials, n (%)	7 (88)	6 (32)	0.01*

\*Fisher exact test; †Wilcoxon rank sum test; ‡Student *t* test.

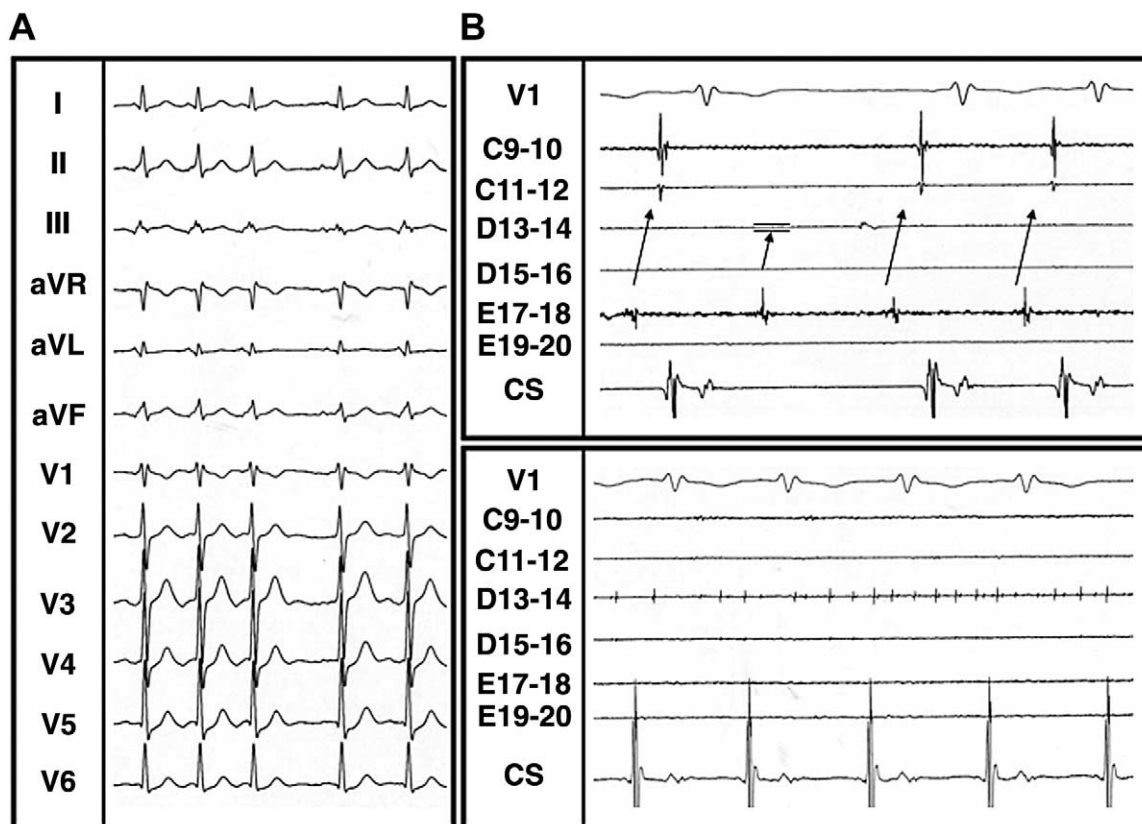
AF = atrial fibrillation; AT = atrial tachycardia; TCL = tachycardia cycle length.

provide a greater density of mapping of AT and to facilitate their three-dimensional localization for ablation. Electro-anatomic mapping may identify critical isthmuses and structural abnormalities that provide the nidus for AT (22,23), but is limited by the requirement for relative temporal stability of the arrhythmia.

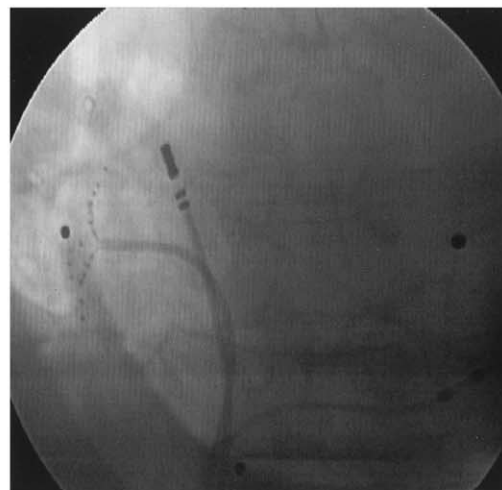
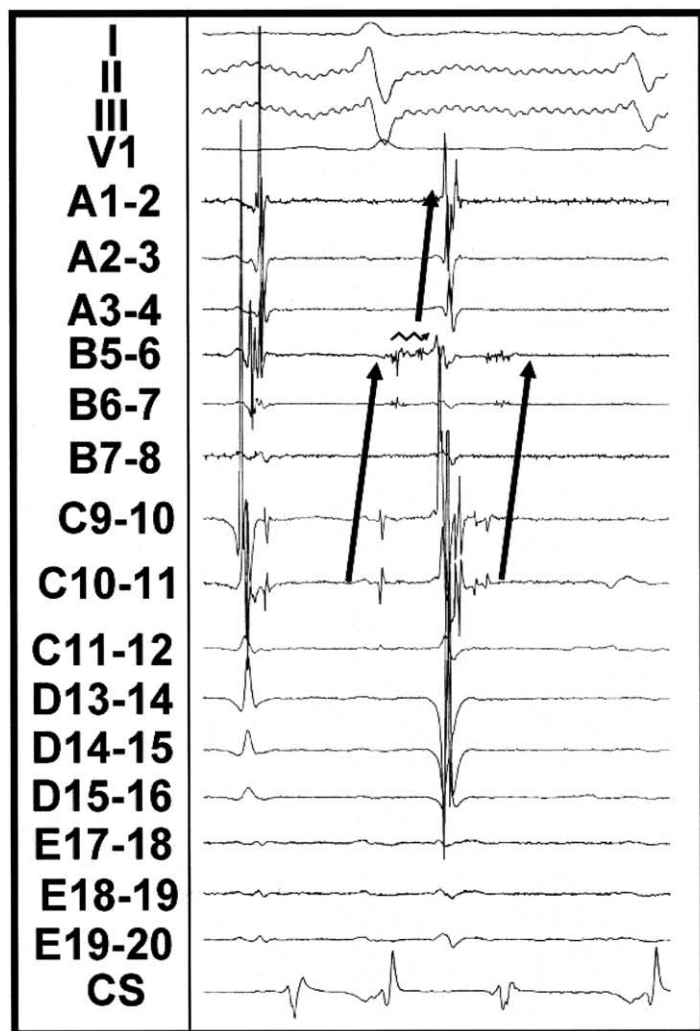
Noncontact mapping provides simultaneous virtual electrograms of the chamber with the advantage of being able to map nonsustained focal AT. It has allowed the recognition of regions of preferential conduction from *origin-to-exit* or

*breakout point* to the remaining atria, as was observed in the current series using contact electrograms (24). However, while providing a global impression of activation, virtual unipolar electrograms are susceptible to errors towards the extremities of the mapping field, and the central array can prove an impediment to navigation of a mapping catheter.

Basket catheter mapping has the ability to perform rapid simultaneous contact mapping of the chamber for the mapping of focal AT (25). While it provides a global density of mapping, its localized resolution is limited. In contrast,



**Figure 7.** (A) Shows the electrocardiogram of an atrial tachycardia with apparent start-stop episodes. (B, top panel) Shows the intracardiac electrograms with the high-density mapping catheter. In this region of atrial electrical silence, two spines record electrical activity. During the pause on the electrocardiogram, activity persists at the origin of the tachycardia on spine-E while there is intra-atrial conduction block that gives rise to the misleading appearance of tachycardia cessation. However, note that the P-wave morphology after the pause is not typical of sinus rhythm. (B, bottom panel) This is also from the same patient and was observed earlier in the mapping process. This recording was observed in the lateral right atrium, again in the midst of an area of electrical silence; spine-D alone demonstrates high-frequency activity. Although surprising that the atria could demonstrate activity at such a rate (mean cycle length 83 ms), this was indeed a sequestered island of activity that was isolated from the remaining atria. Ablation eliminated the local activity but did not change tachycardia. CS = coronary sinus.



**Figure 8.** High-density mapping of a tachycardia focus along the crista terminalis demonstrating the phenomenon of preferential conduction from origin-to-exit or breakout point to the remaining atria. This occurred in a 46-year-old man who presented with atrial tachycardia after having undergone pulmonary vein isolation, left atrial linear ablation, and cavotricuspid isthmus ablation for permanent atrial fibrillation (the crista terminalis had not previously been targeted by ablation). There is reversal of activation of potential on spine-C with the ectopic beat, which then spreads to the adjacent spine-B. At B5–6 continuous activity is observed followed by activation of the remaining atria. A second concealed beat is seen to arise from the origin (C10–11) and traverses to the exit point at B5–6. At this site conduction block is observed to the remaining atria. With longer coupling intervals, such ectopy was observed to excite the whole atria. CS = coronary sinus.

the catheter utilized in the current study allows splaying of the spines against the endocardial surface to achieve high-density contact mapping to accurately localize and characterize the origin of focal ATs.

**Clinical implications.** The ability to position multiple electrodes in a circumscribed part of the endocardium is particularly advantageous for use within the LA and for mapping complex focal AT. In this setting, the catheter facilitated the rapid identification and ablation of focal AT without complications. It is likely that a similar utility would be observed in patients undergoing *de novo* ablation of AT and potentially for atrial sources of activity that may initiate and maintain AF.

Finally, the differentiation of focal from re-entrant mechanisms and documenting their electrocardiographic and electrophysiologic features may facilitate a better under-

standing of these conditions and allow evaluation of their electropharmacologic properties.

**Study limitations.** While this study performed detailed high-density mapping to localize and characterize the origin of AT, it was not designed to reproduce the electropharmacologic properties of these arrhythmias that have been explored previously (1).

**Conclusions.** High-density localized contact mapping facilitates the rapid identification of the origin of complex AT. Characterization of their origin demonstrates that some focal tachycardias can result from localized atrial re-entry.

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